GENETIC RESISTANCE TO ALOMAE-BOBONE VIRUS COMPLEX, THE LETHAL DISEASE OF TARO (*COLOCASIA ESCULENTA* (L.) SCHOTT)

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Abstract

Alomae-Bobone Virus Complex (ABVC) remains the most destructive and the most serious disease of taro *(Colocasia esculenta* (L.) Schott) in the Solomon Islands, and its spread could be a major threat to taro cultivation in the South Pacific region, especially for countries where taro is a major export crop.

To create resistant varieties, a recurrent selection method was chosen and 350 genetic recombinations were performed. The offspring generation was tested for resistance. The performance and reaction of the genotypes to the lethal viral disease was observed during a period covering ten months. The genotypes showed great differences in resistance/susceptibility. Plants with higher levels of resistance were selected for further recombinations. The selected materials (genotypes with higher levels of field resistance) were predominantly those where one of the grandparents was wild taro from areas affected by ABVC.

Introduction

The Alomae-Bobone Virus Complex (ABVC) is most probably a very old disease. It is present in the Solomon Islands where the majority of taro cultivation is found (Malaita, Choiseul, San Cristobal, Santa Ysabel, Kolobangara, and North New Georgia).

In these islands, the growers had several traditional phytosanitary measures to prevent the disease from spreading.

The increase in population pressure on land and a tendency to not put too much value on these traditional practices has resulted in the very rapid spread of these diseases to a point where it is not possible to stop it. Malaita is an example of a high population area, and the disease is more severe in this island than anywhere else. Many taro varieties have disappeared completely, while, there are still tolerant female varieties such as *Mudimudi* and *Oga*.

The work now being carried out is to investigate whether genetic resistance to ABVC exists.

Description of the Problem

ABVC is usually separated into two diseases by farmers. Plants showing Alomae symptoms are characterized by a general progressive necrosis which decreases the size of the plant and leaves, leading to the eventual death of the plant. Young leaves are often pale yellow to yellow in color and crinkled. These leaves do not open normally or when they do they are generally very small in size. Leaf laminas are more pronounced and the veins show symptoms of hypertrophy. In brief, Alomae-designated affected plants give an impression of progressively decreasing in size, at the end of which they disappear completely. The corms, however, remain intact, and if already matured when the disease affects them, the eating quality of the corm is not affected.

Bobone is similar and is usually connected with Alomae. Bobone infected plants are often stunted, with curled and twisted leaves. The plants also have raised or depressed deformations on the leaf petioles (Fig 3). It is important to note that symptoms of Bobone could also be completely different, suggesting that virus complex structure could be more complicated than originally thought. Different types of Bobone symptoms are very distinct on small plants (seedling stage).

According to susceptibility of taro plants to this viral complex, Solomon Islanders divide their cultivars into two groups. These are male and female taros. This division of taros has no connection with the sex expression of the taro plants. Of the four hundred or so varieties grown throughout the country, the majority are male taros.

As a general rule, male taros are larger, produce better tasting and larger corms, and are very susceptible to Alomae and Bobone. When infected with Alomae, the traditional male taros eventually die from the disease.

Female taros are smaller in size and have multiple or a large number of suckers. They yield a large number of small corms. In their reaction to the infection by the virus complex, female taros show symptoms of Bobone but not that of Alomae.

Early Research Work

Alomae-Bobone disease was probably a very old disease associated with taro and traditionally was controlled by cultural practices in which sanitation may have played a major part. The first description of this disease was done in the early fifties (Magee 1954). More detailed analysis, however, was completed much later in the seventies (Gollifer and Brown 1972, Gollifer et al. 1975). At this time, the virus complex was separated into two distinct diseases and was described as the most serious of all taro diseases present in Solomon Islands.

Cytogenetic studies were carried out later (Gollifer et al. 1975) to look at the chromosome number of the male- and female-grouped taros. In this early work, the study showed that female taros had 2n = 28 chromosomes and the male taros had 2n = 42 chromosomes.

Screening of the Solomon Islands' available cultivars (Gollifer et al. 1978) showed that none were resistant.

Following these investigations, Jackson (1981) made an attempt to create resistant varieties through breeding. The results of this work were not positive and gave no promising prospects. All hybrids were destroyed by Alomae when field tested.

In literature, no data can be found on the genetics of resistance to ABVC. In genetic research, taro is a relatively new crop. More work and detailed studies are needed to understand the viruses and the genetics of resistance and/or tolerance to these viruses.

Current Research Work

The question of the genetic resistance to ABVC has been discussed in the Solomon Islands several times. For the third time, the taro breeding program is making a very serious attempt to create resistant or at least tolerant taro genotypes to ABVC.

Within the Ministry of Agriculture and Lands, there is an intensive taro breeding program funded by the Food and Agriculture Organization and the United Nations Development Program, which is based on the recurrent selection method, aimed at accumulating resistant genes that may be present in tolerant female varieties, wild genotypes, genotypes treated by mutagens, and other related species. The central problem in this work is resistance and/or tolerance to ABVC.

However, there are two other important diseases (i.e., taro leaf blight caused by *Phytophthora colocasiae* and *mitimiti* disease of the corm caused by the nematode *Hirschmanniella miticausa*) and several other agronomic characteristics which are included in the breeding program.

Methodology

The breeding program is based on large segregating populations. The basis for this program started in 1989, with observations and studies of natural resistance among existing taro varieties and wild populations of *Colocasia esculenta.*

The preliminary results obtained from this work were very promising. It was clearly established that female varieties could be a good source of resistance for the breeding program. Of the wild populations of *Colocasia esculenta*, two (more or less) uniform populations (groups, types) of *Tiko* Red and White were of great interest. These two types of taros grow along creeks and streams and in swampy or very wet conditions. Observation showed that although when found very close to affected gardens and areas with an abundance of the vector *(Tarophagus proserpina)* but still inside the optimal environment, they do not show symptoms of either Alomae or Bobone. When planted in normal dryland, the symptoms of ABVC do appear. ABVC is predominantly transmitted by *Tarophagus proserpina*, but also by aphids and possibly by several other vectors. The Red *Tiko* is resistant to the *mitimiti* disease.

For creating the first population cycle, more than 350 genetic combinations were made using 250 different genotypes. The parental material included male and female taros, wild *Colocasia esculenta* from different locations, hybrids created from the previous breeding work, and cultivars from overseas. A sample of seed from each cross (the average total number of seeds per cross is approximately 1,000) was planted in the greenhouse, later transplanted outside under shade cloth, and finally planted in Kolobangara (Location 1), Ontong Java (Location 2), Fote (Location 3), and at Dodo Creek Research Station (Location 4).

Locations 1 and 2 were testing the hybrids for resistance to *mitimiti* disease and leaf blight (*Phytophthora colocasiae*), Location 3 was screening for resistance to ABVC and taro leaf blight resistance, and Location 4 was screening for resistance to *Phytophthora colocasiae* and making detailed studies for other important agronomic characters.

The total number of hybrids tested for ABVC in Location 3 was 2,279. The plants were planted successively in seven plots according to the available plant material released from the nursery.

The first plot which is analyzed in this paper was planted in January 1991. In this plot there were a total of 264 plants with spacing of 1.00 m x 0.50 m, making the plot perfectly uniform. The area in which this work was carried out had the optimum soil requirements for taro

Table 1. Crosses tested and analyzed for resistance to Alomae and Bobone.

		Plants within row											
	Rows	1	2	3	4	5	6	7	8	9	10	11	12
F	1	A18 x NP	*	N4 x IA-1	N4 x 7A	N13 x N4A	GA x TKO	N4 x 7A	N13 x D28	N4 x 8A	A18 x N1	TKO x B59	NN**
O	2	N4 x N4A	0	N4 x IA-1	N4 x N1	N4 x N7A				N13 x N4A	N4 x IA-1	TKO x N13	N4B x N3
T	3	N4 x N4A		N4 x IA-1	N4 x N3	A16 x N2	18A x N1	N4B x N3		N4 x 7A			
E	4		N13 xN4A		N6 x N3	N1 x IA-1			A18 x N1	A18 x B81		N4 x IA-1	TKO x B59
	5	N4 x N7A				N14 x 7A	N4 x 7A		TKO x B59	GA x TKO	N9 x N5		N4 x IA-1
F	6	N48 x N14	N5 x D22		TKO x N1	NN**	N4 x IA-1			A18 x N1	N4B x N3	GA x TKO	
E	7	TKO x N5		12A x N4A	N1 x 48	TKO x B59	A18 x B81	N1 x N5		N13 x ?	N4 x 7A		N4 x N4A
S	8	TKO X N13		N4 x IA-1		A18 x B81	E28 x N9	N1 x N5	N5 x D22	N1 x 60B	N13 x N4	N13 x D28	N48 x N14
	9	N13 x D28			N1 x 18A	N14 x IA-1		TKO x N13	GA x N3	N13 x N9A			
M	10	N4 X N3	N7 X N7A		N4 x 7A				A13 xN3	TKO x A1	N4 x N4A		
a	11	TKO x ?			A18 X B81			TKO x A13		N5 x N4		TKO x N13	
i	12	N4 x N4A		N15 x 7A	GA x TKO		A18 x B81		CH5x SW1		E28 X N5	GA x TKO	
a	13	N1 x N5		N1 x N4	N15 x 7A	N1 x A18	N4 x 7A		N1 x N5	A18 x N3	N1 x N5		A18 x B81
i	14	N5 x N5A		N4 x IA-1	N1 x N5	N5 x N3		TKO x N13		N3 x N4		N4 x N4A	
t	15	N5 x N4	N9 x N4		N9 x B28	N7A x N5		A18 x B81		E 28 x N9		TKO x N14	
a	16	12A x N4A		N6 x N3		GA x N8		N4 x N4A		N7 x N4A	A18 x N4		
	17	TKO x B59	N28 x N9	N5 x N4	N1 x N5	E32 x N5	N3 x D28	N1 x N4	N1 x N5		N4 x N4A	N5 x N4	N7 x N4
P	18	A13 x B31		N4 x 7A				A18 x N1		N1 x N4	N9 x B28	N4 x IA-1	A16 x N3
L	19	TKO x A1	N9 x N4		N3 x N4A	N5 x N4	N1 x 60B	N4 x 7A	N3 x N4	CROSS 1	N4 x N14	NN**	
O	20	N5 x N4		N6 x N4				N1 x 60B		N4 x N4A		TKO x B59	TKO x N3
T	21	A16 X N4		N1 x N4			N1 x N5		N3 x D28		N9 x B28	TKO x B59	
	22	N13 x D28		N1 X 10B	N15 x 7A	N5 x N8		N15 x 7A	N9 x D28		N0 x D28	TKO x N13	18A X N1
1													

Planting date 24/1/91

* = plant 2 the same as previous plant; ** = label lost; empty space = plant from the same cross as previous plant
distance between rows = 1.0 m; distance within rows = 0.5 m

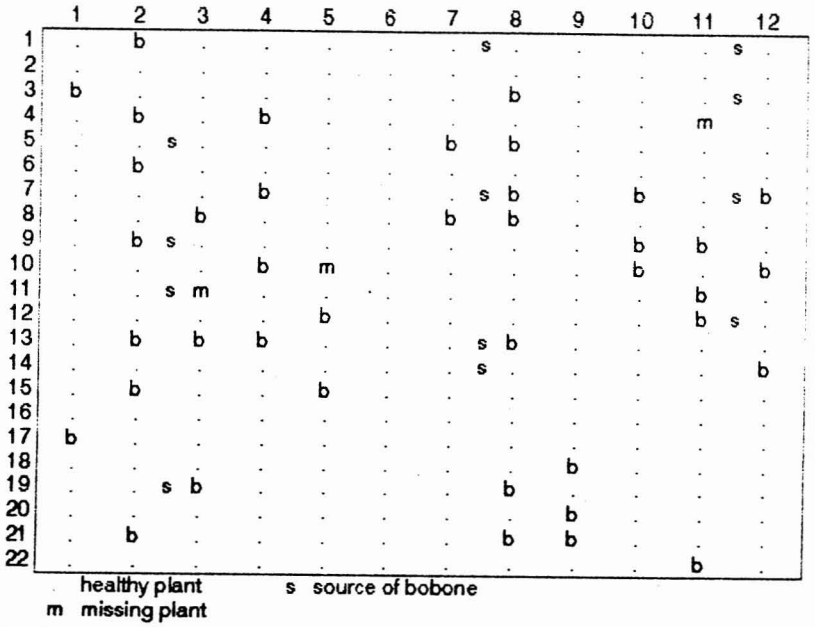

Fig. 1. First plants showing Bobone (b). (. = healthy plant; s = source of Bobone; m = missing plant)

cultivation. Each of the 264 hybrids in plot 1 were planted in a way which ensured that data recorded could be related back to the parents of each cross (Table 1).

Virus disease was introduced into the plot by planting infected materials among the hybrid plants (Fig. 1). These infected planting materials were collected from farmers' gardens. Taro leafhopper (*Tarophagus proserpina*) was already present. The work started at the beginning of the rainy season.

The detailed assessments of infection were carried out seven times over a growing period of ten months.

Results

The first Bobone-associated symptoms within the plot appeared three weeks after planting. These were on plants (hybrids) which were closest to virus source plants (Fig. 1).

During the first three months, the ABVC spread rapidly, decreasing the number of healthy plants drastically (Fig. 2, Table 2). In the fourth month and the following months, several plants recovered from the disease symptoms and were quite healthy. These were the plants which had had the typical Bobone symptoms before. All the old infected leaves disappeared, the new growth was symptomless and very clean. This, therefore, increased the number of healthy plants. In June, the disease incidence increased again and the number of healthy plants decreased for a second time. In this period, some of the hybrids were showing symptoms of Bobone for the second time and some for the first time.

In July, the first significant number of plants with Alomae symptoms were observed. The disease spread quite rapidly until almost every plant was infected by October (ten months after planting) (Fig. 2, Table 2). The last assessment carried out in December showed that all the aboveground parts of the majority of hybrids had died.

The cultivars used as parental materials had their average growth cycle (from planting to harvest) of five to six months. The growth cycles of the two wild *Tiki* populations were even much shorter, especially when planted in dryland conditions. These were fully matured after four months of growth.

From the final screening, 49 plants were selected out of a population of 2,279 hybrids. Twelve plants were later destroyed as they developed Bobone symptoms.

Discussion

This preliminary investigation indicates that there is definitely genetic difference in resistance to the virus complex Alomae-Bobone. The differences in levels of resistance described in this work suggest that it is probably strongly influenced by age of the plants and environmental conditions. The Alomae virus complex (i.e., virus particles) probably has a very low rate/capacity of building up to levels to cause a disease and produce a visible symptom and is probably different for each genotype. This, too, could be greatly influenced by environmental conditions and health status of the plant.

The hybrids approaching maturity were probably more susceptible to viral infection of the Alomae type. It is suggested from this work that this may be due to a build-up of levels of the virus particles within the plant and the general senescing of plants as they mature. Senescing plants may allow for optimum conditions which are suitable for rapid multiplication of the virus particles of the viral complex.

Analysis of the hybrids that showed a higher degree of resistance to the disease did not give any conclusive indication of which combination was the best. It was assumed at the beginning of the work that combinations between wild taros (from Malaita) and normal cultivated taros would probably be the best. However, the analysis of pedigree (parentage) showed that there were great differences in all crosses. The materials selected for further crossings following the results of this investigation were predominantly those where one of the grandparents was wild material (Table 2).

This investigation indicates that larger populations need to be screened and that the genetic mechanism of taro resistance to ABVC is probably very complicated. The total number of viruses and the viral particles involved or the variations within each particle in the complex still has to be sorted out and determined.

Because of the large number of populations which need to be tested, the second-cycle population genotypes are being screened in small cages and at an earlier stage of growth. This is from the fourth to eighth leaf stages and about 1,000 seedlings at a time. Infected plants as well as insect vector originating from Location 3 were introduced into each cage along with the seedlings. It is hoped that in this way more detailed studies of the development of the disease can be carried out on a large number of screened hybrids before the selected hybrids are put through field screening for final selection.

The virulence of the disease is very high. *Alocasia macorrhiza* (wild) found adjacent to the screening area also showed typical symptoms of Bobone (Fig 3). Similar symptoms were also observed in *Xanthosoms sagittifolium* and *Cyrtosperma chamissonis*.

The resistance is probably controlled by several genes. The total number of virus particles involved in the complex, their virulence, and the relationship with the corresponding resistance/tolerance genes are yet to be discovered. Through recurrent selection, it will be possible to accumulate these genes in the population and eventually create resistant or at least more tolerant varieties after a certain number of cycles of selection. If the resistance and/or tolerance is connected with the growth period, the selection for early maturing genotypes might also result with higher tolerance due to disease escape. This could be one possible partial and/or short-term temporary solution.

Table 2. The dynamics of the development of Alomae-Bobone virus complex symptoms.

Plants within row

	1							2							3							4							5							6						
1	H	H	H	S	–	–	–	B	R	H	H	S	–	–	H	H	B	B	M	–	–	H	H	H	H	B	A	A	H	B	A	A	A	M	–	H	H	H	H	H	A	A
2	H	H	H	H	H	B	A	H	B	H	H	H	A	A	H	H	B	H	H	A	A	H	B	B	H	B	B	A	H	H	H	H	S	–	–	H	H	B	B	B	M	–
3	B	B	R	B	B	A	A	H	B	R	B	M	–	–	H	H	B	B	B	B	A	H	B	R	B	R	A	A	H	H	B	R	H	A	A	H	B	R	R	B	A	A
4	H	H	B	B	R	B	A	B	B	R	H	S	–	–	H	B	R	H	M	–	–	B	R	R	H	S	–	–	H	B	B	H	B	A	A	H	H	B	H	B	A	A
5	H	H	B	B	B	A	A	H	H	B	B	R	B	A	H	H	B	H	B	A	A	H	H	B	B	A	A	A	H	B	B	B	B	B	A	H	B	B	B	B	B	A
6	H	H	B	R	B	A	A	B	B	B	R	B	B	A	H	B	B	R	R	A	A	H	H	B	B	B	M	–	H	H	B	R	B	A	A	H	H	B	B	B	A	A
7	H	H	B	R	H	B	A	H	H	B	B	R	A	A	H	H	B	R	R	A	A	B	R	B	B	B	R	A	H	B	B	B	B	A	A	H	H	B	B	B	A	A
8	H	H	B	R	B	A	A	H	H	B	B	B	A	A	B	B	B	H	B	B	A	H	B	M	–	–	–	–	H	B	B	R	B	A	A	H	B	B	B	B	A	A
9	H	H	B	B	B	A	A	B	B	B	H	S	–	–	H	B	B	B	B	B	A	H	H	B	B	M	–	–	H	H	B	H	S	–	–	H	H	B	R	B	A	A
10	H	H	B	R	H	B	A	H	B	B	H	H	B	A	H	B	B	H	B	B	A	B	R	B	H	B	A	A	M	–	–	–	–	–	–	H	B	B	R	B	A	A
11	H	H	B	R	H	B	A	H	B	H	H	S	–	–	M	–	–	–	–	–	–	H	H	H	H	S	–	–	H	H	B	H	B	A	A	H	B	B	B	R	A	A
12	H	H	B	H	H	A	A	H	B	B	H	H	B	A	H	H	B	H	H	B	A	H	B	B	B	B	A	A	B	R	B	B	B	B	A	H	B	H	H	S	–	–
13	H	H	B	B	H	A	A	B	R	B	R	B	A	A	B	B	B	H	H	A	A	B	B	B	B	B	A	A	H	B	B	H	S	–	–	H	H	B	B	B	A	A
14	H	H	B	B	B	A	A	H	B	B	B	H	A	A	H	H	H	H	S	–	–	H	H	B	B	H	A	A	H	B	H	R	B	A	A	H	I	B	B	B	A	A
15	H	H	B	H	B	A	A	B	B	B	R	H	A	A	H	B	B	B	R	B	A	H	B	H	B	B	B	A	B	B	B	B	B	A	A	H	B	B	B	R	A	A
16	B	B	B	B	R	B	A	H	B	B	R	B	B	A	H	B	B	R	B	A	A	H	H	B	B	B	A	A	H	H	B	R	B	A	A	H	H	B	H	H	A	A
17	B	B	B	B	R	A	A	H	B	B	R	R	A	A	H	B	H	B	B	B	A	H	B	M	–	–	–	–	H	H	B	R	B	A	A	H	H	B	H	B	A	A
18	H	B	B	B	H	S	–	H	B	B	R	H	A	M	H	B	R	B	R	A	A	H	H	B	B	B	R	A	H	H	B	B	B	B	A	H	H	B	B	R	A	A
19	H	B	B	H	H	B	A	H	B	B	R	H	A	A	B	B	B	B	R	A	A	H	H	B	B	M	–	–	H	B	B	R	H	B	A	H	H	B	R	H	A	A
20	H	B	B	R	B	A	A	H	B	B	R	R	A	A	H	B	M	–	–	–	–	H	B	B	B	R	A	A	H	B	B	B	R	B	A	H	B	B	B	B	A	A
21	H	H	H	H	H	S	–	B	B	B	R	H	A	M	H	B	B	B	R	A	A	H	B	B	B	R	A	A	H	B	B	B	R	A	A	H	B	B	B	R	A	A
22	H	B	B	H	H	A	A	H	B	B	B	B	A	A	H	B	B	B	B	A	A	H	B	R	R	R	B	A	H	B	M	–	–	–	–	H	B	B	R	R	A	A
	1*	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7

Plants within row

	7							8							9							10							11							12						
1	H	H	H	H	H	A	A	H	B	H	H	H	B	A	H	H	B	R	H	B	A	H	H	H	H	H	A	A	H	B	H	H	H	A	A	H	B	R	H	H	A	A
2	H	R	B	H	H	B	A	H	M	–	–	–	–	–	H	B	B	B	R	A	A	H	H	H	H	H	A	A	H	B	H	B	B	A	A	H	H	B	R	H	B	A
3	H	B	R	B	R	A	A	B	R	R	H	H	B	A	H	H	B	B	B	A	A	H	H	B	B	R	A	A	H	H	H	H	S	–	–	H	B	H	B	M	–	–
4	H	H	B	B	B	A	A	H	H	B	B	B	M	–	H	H	H	H	H	A	A	H	H	B	B	B	A	A	M	–	–	–	–	–	–	H	B	H	B	B	A	A
5	B	R	B	B	A	A	A	B	R	B	B	B	A	A	H	H	B	B	R	A	A	H	H	H	H	B	A	A	H	H	H	H	B	A	A	H	H	B	H	B	A	A
6	H	H	B	B	B	A	A	H	H	B	B	A	A	A	H	H	B	B	B	A	A	H	H	B	H	H	B	A	H	H	B	B	B	A	A	H	H	H	H	B	A	A
7	H	H	B	B	B	A	A	B	B	B	H	H	B	A	H	B	B	B	B	M	–	B	B	B	R	B	A	A	H	H	B	H	B	A	A	B	B	B	B	R	A	A
8	B	R	B	H	B	A	A	B	B	B	H	B	B	A	H	H	M		–	–	–	H	H	B	B	B	A	A	H	H	B	R	B	A	A	B	R	B	R	R	A	A
9	H	B	B	B	B	A	A	H	B	B	R	B	A	A	H	H	B	H	B	R	R	B	B	B	B	B	B	A	B	R	H	H	S	–	–	H	H	B	H	B	A	A
10	H	B	B	A	A	A	A	H	B	H	H	B	A	A	H	H	B	R	B	A	A	B	H	B	H	B	A	A	H	H	B	H	S	–	–	B	H	B	B	B	A	A
11	H	H	B	H	B	B	A	H	B	B	B	B	A	A	H	H	B	B	R	S	–	H	A	A	A*	B	A	A	B	H	B	H	B	A	A	H	B	B	H	B	A	A
12	H	B	B	R	R	A	A	H	B	B	R	R	A	A	H	H	B	R	B	B	A	H	H	H	H	H	B	A	B	R	B	H	H	B	A	H	M	–	–	–	–	–
13	H	B	B	B	B	A	A	B	R	B	B	R	A	A	H	B	B	B	R	A	A	H	H	B	B	R	A	A	H	H	B	B	R	A	A	H	M	–	–	–	–	–
14	H	B	B	H	B	A	A	H	B	B	B	R	S	–	H	B	B	B	B	A	A	H	H	R	H	S	–	–	H	H	B	H	S	–	–	B	H	B	H	A	A	A
15	H	B	B	R	R	A	A	H	H	B	H	H	A	A	H	B	B	B	B	A	A	H	H	B	B	R	A	A	H	B	B	H	B	B	A	H	B	B	B	R	B	A
16	H	H	B	R	R	A	A	H	H	B	B	R	A	A	H	H	B	B	R	A	A	H	H	B	B	R	A	A	H	B	H	B	R	A	A	H	B	H	H	B	A	A
17	H	B	B	H	S	–	–	H	B	B	B	B	A	A	H	H	B	H	S	–	–	H	H	B	B	R	A	A	H	H	H	B	R	A	A	H	B	H	H	S	–	–
18	H	H	B	B	R	A	A	H	H	B	H	H	A	A	B	B	B	H	H	A	A	H	H	B	B	B	B	A	H	B	B	R	R	A	A	H	B	H	H	S	–	–
19	H	B	B	B	R	A	A	B	B	B	B	R	A	A	H	H	B	R	R	A	A	H	B	H	B	R	A	A	H	B	B	H	B	A	A	H	B	B	B	R	R	A
20	H	B	H	H	A	A	A	H	B	B	H	B	A	A	B	B	B	H	B	B	A	H	H	R	H	B	A	A	H	B	B	H	H	S	–	H	H	B	H	H	S	–
21	H	B	H	H	B	A	A	B	R	B	H	B	B	A	B	B	B	H	B	A	A	H	H	R	H	S	–	–	H	B	H	H	B	A	A	H	H	H	H	H	A	A
22	H	B	B	H	H	A	A	H	H	B	H	H	B	A	H	H	H	H	S	–	–	H	H	H	H	S	–	–	B	B	B	H	B	A	A	H	B	B	H	B	B	A
	1*	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7

* Date of assessment
1. March 1 1991
2. March 22
3. April 25
4. May 23
5. July 9
6. August 29
7. October 10

A – Alomae (dying plant)
B – Bobone
H – Healthy plant
I – Taro beetle damage

M – Missing plant
R – Recovered from bobone
S – Selected and removed plant for crossings

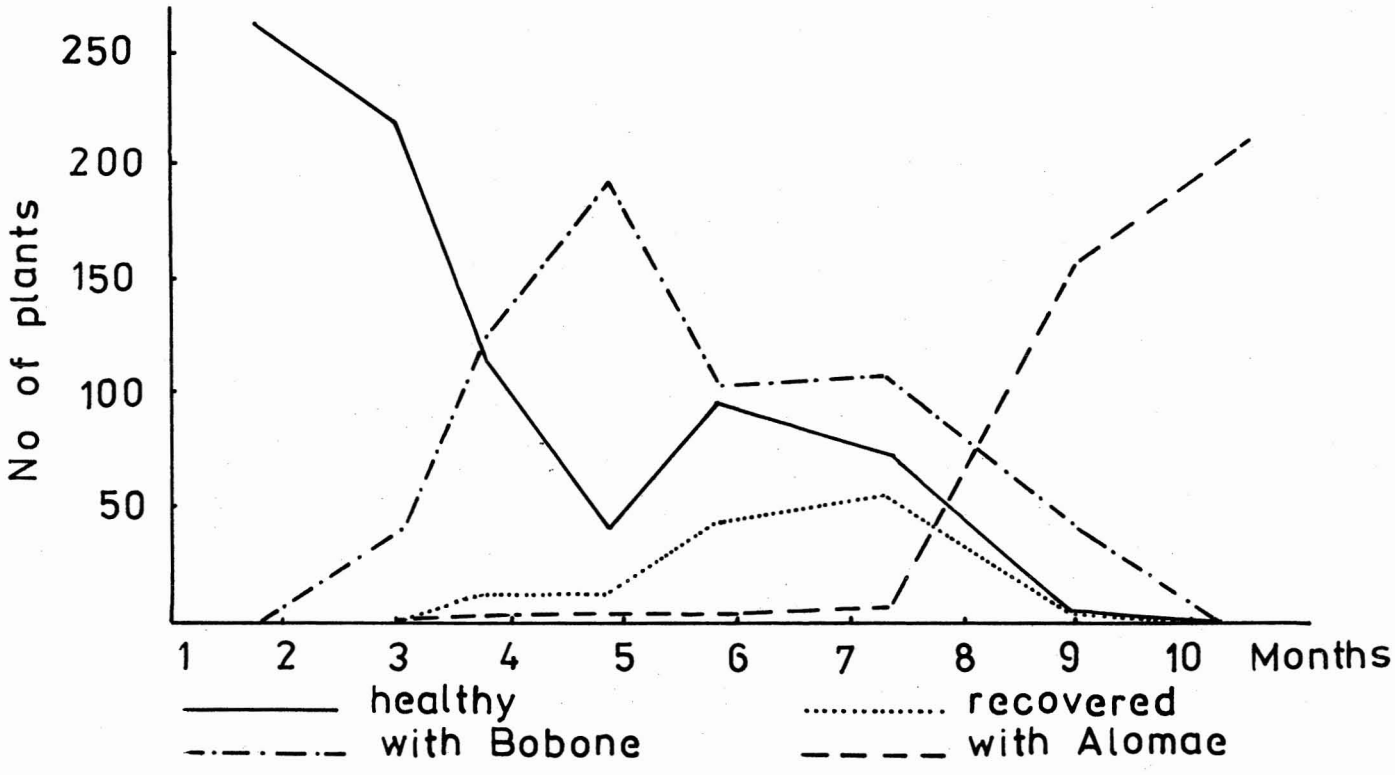

Fig. 2. Dynamics of Alomae-Bobone virus complex development.

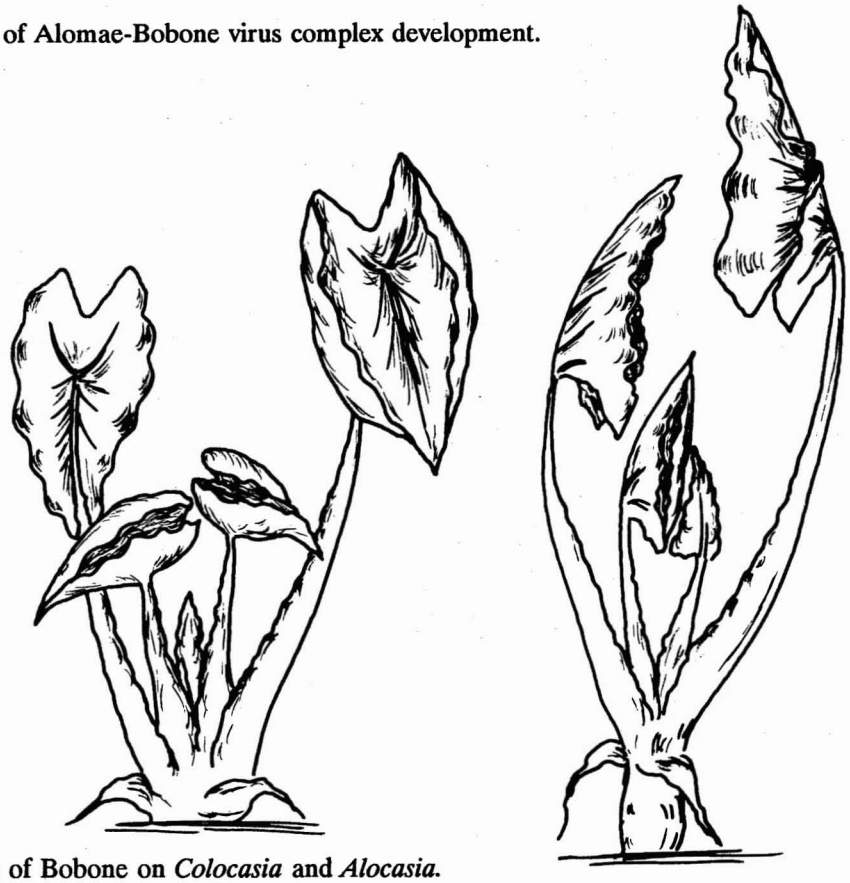

Fig. 3. Symptoms of Bobone on *Colocasia* and *Alocasia.*

Conclusion

ABVC remains the most destructive disease of taro in the Solomon Islands.

In this investigation, plants with the symptoms of Bobone appeared three weeks after planting. The number of these Bobone-affected plants increased rapidly during the first three months after planting. At that time, genotypes showing certain level of resistance recovered from the disease. Some of these genotypes showed the symptoms of Bobone for a second time after having recovered from the first infection.

The number of healthy plants decreased rapidly at the beginning. As plants recovered after the first three months, the number of healthy plants or symptomless plants increased slightly. Healthy, resistant plants were selected for further crossings.

The first significant number of plants showing the lethal Alomae symptoms appeared in July, six months after planting. Ten months after planting, almost all the plants were dead.

These findings indicate that besides genetic structure the resistance to the virus complex is probably related to plant age, virus particle accumulation, and environmental conditions (stress).

The Alomae symptoms became very distinct at the end of the rainy season.

The selected genotypes for the next selection were those with a higher level of resistance and predominantly from combinations where one of the grandparents was a wild genotype from the areas affected by the lethal disease.

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